Bandolier 39

What do we think?
What do we know?
What can we prove?

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Frogs in a jam jar, 1997

We sit like frogs in a jam jar, ready to jump as the next bit of information, mostly paper, rains down on us, and agonising about when to jump, and how high. The average UK medical mortal spends about 30 minutes a week reading journals, so we are always terrified of being behind. Filtering the volume of paper (estimated at about 3 kilos/week for the average GP) itself takes up 30 minutes. The twin problems are how to get the right stuff (evidence on which we should act) and how do we get that evidence into practice.

The right stuff

Patients hear on TV that there is a new whizzo remedy for something. They hear it before you do and come and ask about it. You know nothing (just a few weeks behind with the reading and no time to watch TV). A document in the 3 kilos six months or a year from now is six months or a year too late!

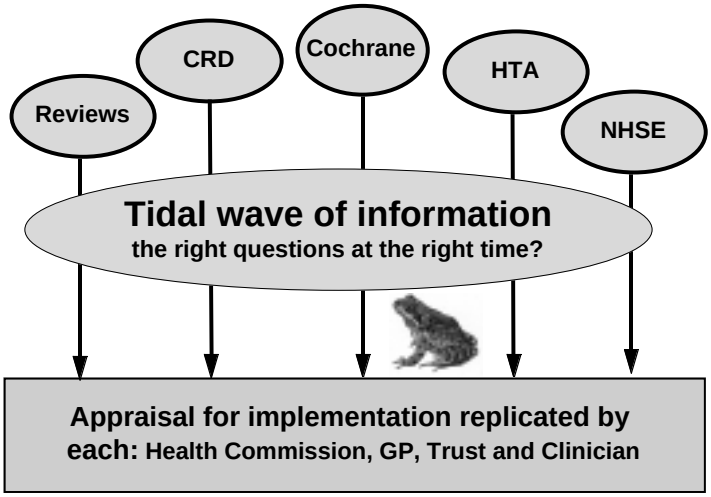
The process of working out how the NHS will deal with this advance takes place in every health authority, Trust, or GP surgery. While ***Bandolier*** can understand the political imperative of devolving management decisions as much as possible, it makes little sense for every health authority, Trust, or GP to go through the same appraisal process, at huge cost. We need a new mechanism to help us. Perhaps this is at the heart of the implementation gap - what is stopping evidence getting into practice - spotted by Walshe & Ham below.

Acting on the evidence

This is the title of a superb research paper from Kieran Walshe and Chris Ham of the Health Services Management Centre at the University of Birmingham. It presents the findings of a study to assess the progress of evidence-based health care in the NHS and to identify innovations and approaches which might serve as models of good practice. ***Bandolier*** thinks they have done a good job. There are some thoughtful words which should be read by those responsible for management of Health Authorities and Trusts, and by policy makers. While primary care was not a subject of the research, many of the conclusions and suggestions are immediately applicable to primary care.

Making it happen

Perhaps the most important issue facing the health service is not how it should be organised or financed, but whether the care it provides actually works. This applies not only to particular interventions, but to whole packages of care, and one of the features of the evidence-based approach - systematic



review - has been to starkly demonstrate that what we *think*, or what we believe we *know*, falls flat when it comes to *proof*.

The problem is one of handling the tidal wave of knowledge coming our way. High quality evidence is increasingly available, not just through the Cochrane Collaboration, NHS Centre for Reviews and Dissemination, or Health Technology Evaluation (all parts of the NHS R&D initiative), but through the efforts of researchers all over the world producing systematic reviews published in academic medical journals.

Impact and impact factors

Walshe & Ham point to the "curious dissociation between the research and development process and the world of clinical practice". If research agendas are being set by academics and funding bodies, we should not be surprised. Academics (or at least those who make their living in Universities) live and die by academic brownie points - which come from individuals and departments publishing results of research in journals with the highest "impact factors", and whether or not they change practice is irrelevant.

The fact that most practitioners, let alone managers, will never

In this issue

see the journal is neither here nor there - impact (by which we mean the information getting to the largest number of potential users) will be subordinated to the next grant application, and driven by publishing in the BMJ, Lancet, JAMA, Annals or New England Journal. The whole raison d'être of ***Bandolier*** is to be a signpost to good quality information, published where you might not find it.

Managing knowledge

We need a few new developments. It seems to ***Bandolier*** that health authorities and trusts are going to need knowledge officers to handle the tidal wave and to direct knowledge at a local level. Nationally we need an information exchange - so that some of the excellent implementation examples described by Walshe & Ham, or coming from the King's Fund *PACE* programme, or being developed locally in many health authorities and Trusts don't need to be re-invented.

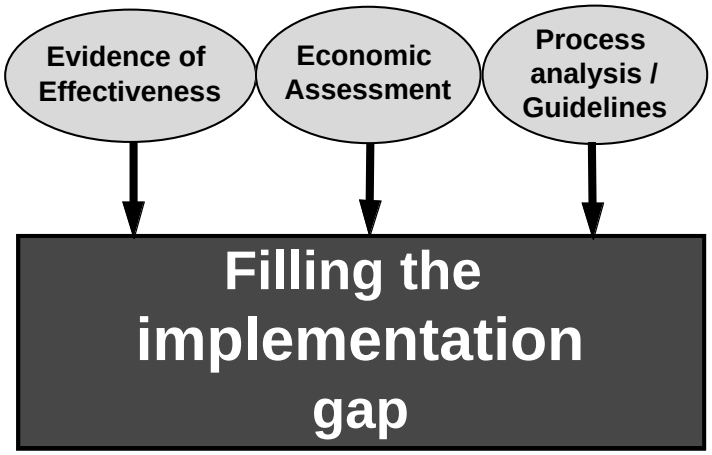
And where's the beef?

In his foreword to the second edition of the ***Bandolier*** Annual, Professor Richard Lilford takes ***Bandolier*** to task for not making more effort to cover modelling, patient preference issues, and issues of cost utility and decision or process analysis. Other readers make the same criticism. We agree with that, and would love to see more information on costs and economics based on sound evidence. Perhaps ***Bandolier*** is looking in the wrong places, but we don't find much available to fill information and implementation gaps.

Filling the implementation gap

Walshe & Ham point to a gap between the production of knowledge and its implementation. ***Bandolier*** agrees that such a gap exists, and has pointed out the irony of spending large amounts of money on technology assessment, for instance, and then printing only 100 copies of reports at £50 a time (***Bandolier*** 37). But the gap will not be filled by researchers whose prime interests lie elsewhere, and it is inefficient to expect every gap to be filled in each locality.

We need a national implementation programme which can create task forces to do the work to bridge the gap. These task forces should include researchers, health economists, specialists, GPs, public health doctors and managers, and would aim to fill the implementation gap by summarising the evidence on effectiveness, exploring the heath economic arguments, and examining the process of delivering the highest quality effective healthcare with pro-forma guidelines which can be adapted to local needs. Such implementation task forces could not only look at existing services or treatments, but also should be expected to examine emerging trends (like viral load tests and protease inhibitors for HIV, for instance).



We should thank Walshe & Ham for concentrating our minds on how we deliver a better standard of effective service. ***Bandolier*** recommends reading their monograph, which is available from the NHS Confederation Publications office (Fax +44 (0) 121 414 1120).

Antibiotics do not prevent infection in simple wounds

The team that brought us the analysis of antibiotics in dog bites (***Bandolier*** 16) has also produced a meta-analysis of the use of antibiotics in simple non-bite wounds [1].

Inclusion criteria

The search was for randomised trials of prophylactic systemic antibiotics for non-bite wounds managed in emergency departments. The date of the last search was December 1993. Studies were included if the authors stated they had randomly assigned patients with uninfected wounds to an antibiotic treatment or a control group, and then followed patients to see if the wound became infected.

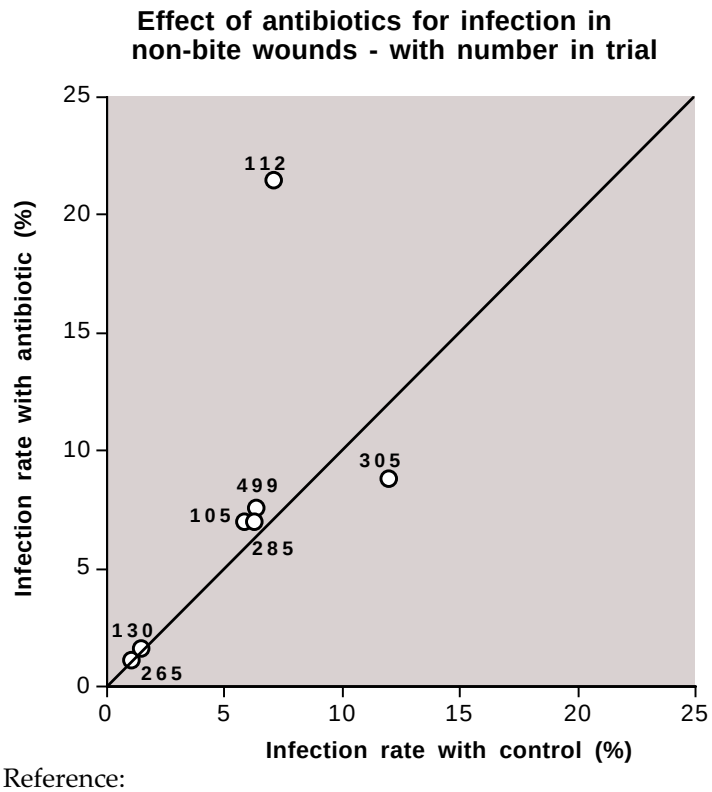
Nine studies were found; two were excluded. The seven included studies had information on 939 patients treated with antibiotics and 762 controls and were published between 1975 and 1983. Five studies were done in the UK and two in the United States. All studies used either cephalosporins or penicillins, some of which were resistant to penicillinase. Five studies were limited to hand wounds, and five to sutured wounds. The nature of the controls in these studies was not clearly described in the meta-analysis.

Results

The rates of follow up in the seven studies were high, between 90 and 95%. The cumulative rate of infection in controls was 1.1% to 12% with a mean of 6%. Patients on antibiotics had a higher rate of infection, with a combined odds ratio of 1.2 (95%CI 0.8 to 1.8). Subgroup analysis did not reveal any groups which had any significant difference from controls, which was hardly surprising since no single study showed any difference between patients treated with systemic antibiotics and control subjects. So in 1,204 patients treated with penicillinase resistant antibiotics the odds ratio was 1.0 (0.6 to 1.7).

Comment

These results are quite negative. There is no evidence that antibiotics prevent infection in simple wounds. After dog bites, antibiotics were effective, with a pooled NNT of 16 (***Bandolier*** 16).



Reference:

1 P Cummings, MA Del Beccaro. Antibiotics to prevent infection of simple wounds: a meta-analysis of randomized studies. American Journal of Emergency Medicine 1995 13: 396-400.

DIAGNOSING DIABETES

Meta-analysis with single patient data is rare, but meta-analysis of a diagnostic test with single patient data is probably unique. It is even more remarkable for diabetes, where in the USA at least half the diabetics are undiagnosed and where only 20% of diagnosed diabetics are diagnosed according to the acknowledged World Health Organisation criteria of fasting plasma glucose and oral glucose tolerance test on at least two occasions. Some brave hearts from Los Angeles have performed a superb analysis to determine that a single haemoglobin A_{1c} test (HbA_{1c}) can diagnose diabetes with confidence. ***Bandolier*** can only bring you a flavour of the enormous amount of work that went into this analysis.

Glycosylated haemoglobin

Glycosylated haemoglobins are normal haemoglobin to which a glucose molecule becomes added in a non-enzymatic manner. The percentage haemoglobin that is glycated is directly proportional to the time that red blood cells have been exposed to glucose, and to glucose concentrations. Measurement of the glycated haemoglobin fraction gives an integrated picture of the average blood glucose concentration during the half life of the cells - that is over the last 60 days. HbA_{1c} is usually given as a percentage of the total haemoglobin.

Data search

The authors searched for papers which had data on both an oral glucose tolerance test and glycosylated haemoglobin. They then contacted authors to try to obtain individual patient data. Out of 34 possible studies, 31 investigators responded, and 18 were able to provide data. Fasting plasma glucose concentrations, two-hour post dextrose glucose concentrations and glycosylated haemoglobin levels were available from 11,276 individuals (83% of all subjects in the literature). Analyses were restricted to 10 studies with HbA_{1c} information on 8,255 individuals.

WHO criteria

The WHO criteria for interpretation of glucose tolerance tests after 75g oral dextrose is:

Normal: Fasting plasma glucose <6.4 mmol/L ***and*** 2 hour plasma glucose <7.8 mmol/L

Diabetes: Fasting plasma glucose ≥7.8 mmol/L ***or*** 2 hour plasma glucose ≥11.1 mmol/L

Impaired glucose tolerance (IGT): any other condition

HbA_{1c}

HBA_{1c} levels in individuals with normal glucose tolerance tests was a mean of 5.3% with a standard deviation of 0.5%. Using the usual practice of describing a normal range as ± 2 standard deviations, the normal range was 4.3% to 6.3%.

However, with some beautiful but complicated modelling the authors were able to define a level of 7.0% as a cutoff for diagnosing diabetes. In their model they identified subpopulations representing normal subjects and diabetics. The 7.0% cutoff gave a sensitivity of 99.6% - that is a HbA_{1c} of 7.0% correctly identified 996 out of 1000 patients who had diabetes in their model. The same cutoff had a specificity of 99.9% - that is a HbA_{1c} of 7.0% correctly identified 999 out of 1000 patients who were normal in their model.

When applied to the actual oral glucose tolerance results, of those subjects with an HbA_{1c} level of at least 7.0%, 89% had diabetes, 7% had impaired glucose tolerance, and 4% were normal.

Comment

The authors give good arguments why HbA_{1c} levels should be used for diagnosing diabetes, including the difficulty of performing glucose tolerance tests reliably, the lack of reproducibility of the oral glucose tolerance test, and the clinical practice of using HbA_{1c} as a surrogate test for glucose lowering, with the aim of keeping the HbA_{1c} levels of diabetic patients below 7.0%. Moreover, an HbA_{1c} level of below 7.0% would generally be treated by diet and exercise, regardless of the diagnosis of impaired glucose tolerance or diabetes defined by an oral glucose tolerance. Perhaps we will learn soon that diabetes can be diagnosed with a fasting plasma glucose plus a single HbA_{1c} level.

Reference:

1 AL Peters, MB Davidson, DL Schriger, V Hasselblad. A clinical approach for the diagnosis of diabetes mellitus: an analysis using glycosylated hemoglobin levels. Journal of the American Medical Association 1996 276: 1246-52

TRANSDERMAL NICOTINE FOR ULCERATIVE COLITIS

Ulcerative colitis has an incidence of 5-10 per 100,000 in the UK. For some time a lower incidence has been noticed in people who smoke cigarettes. A fine case-control study from Chicago put some figures on this, and the *observation* was followed by randomised trials where patients were treated with nicotine to allow us to establish *causation* and the *magnitude* of the effect.

Case-control study

One hundred patients with ulcerative colitis were matched for age and sex with community control subjects, and selected at random for a telephone interview to collect information on smoking habits, race, religion, income, occupation and education [1]. Smoking habits at the onset of symptoms were analysed using logistic regression.

Compared with those who had never smoked, current smokers were much less likely to have ulcerative colitis (odds ratio 0.13, 95% CI 0.05 to 0.38) - that is they were about eight times less likely to have ulcerative colitis. Former smokers' risk was no different from non-smokers.

Transdermal nicotine treatment

Two randomised studies have tried to answer the obvious question - does nicotine help in treating ulcerative colitis?

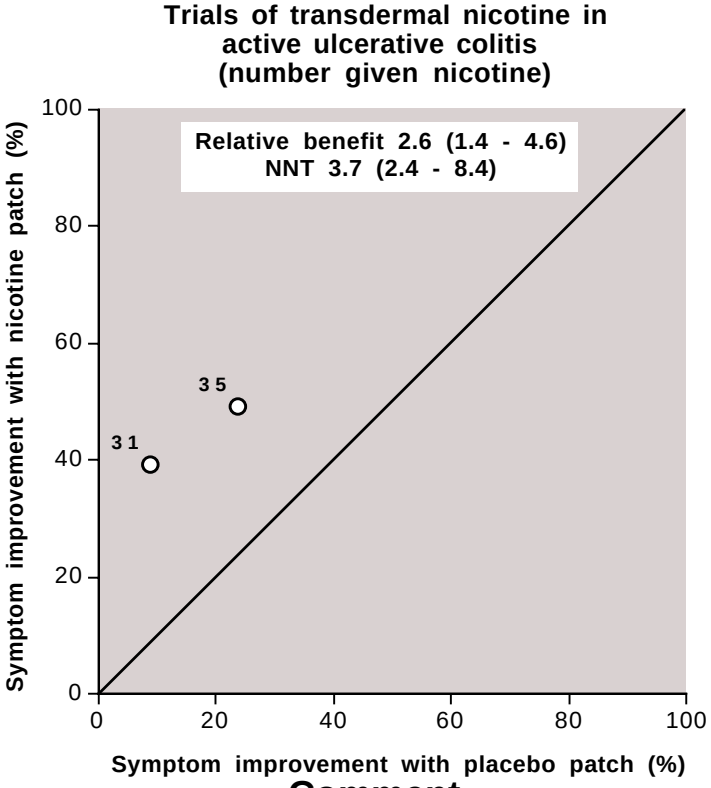
The first study [2], from the UK, randomised 72 patients with active ulcerative colitis between transdermal nicotine patches or placebo patches for six weeks and measured plasma nicotine and cotinine (a metabolite of nicotine). None of the patients currently smoked. The mean patch dose for those given transdermal nicotine patches was 17 mg/day (achieved by balancing adverse effects). For those given placebo patches, the mean daily "dose" was 19 mg ; the study was double-blind.

There were lots of outcome measures, including clinical symptom scoring, sigmoidoscopy and histology. The crude analysis showed that 17/35 patients (49%) given transdermal nicotine had complete symptomatic relief and a global clinical grade of 0 (1-3 bowel motions a day, formed, without blood or mucus, and with no constitutional symptoms) compared with 9/37 patients (24%) given placebo.

These findings have been largely confirmed by a US study [3]. Here 64 non-smoking patients with mildly to moderately active ulcerative colitis were randomised in a double-blind comparison of transdermal nicotine and placebo patches. The daily dose of nicotine was 11 mg for 1 week, increased up to 22 mg/day for three weeks.

The outcome was measured on a disease activity index which included scored for stool frequency, rectal bleeding, sigmoidoscopy findings, physician assessment and histology. A fall of three points on this scale was considered to be a clinical improvement. Such an improvement occurred in 12 of 31 patients (39%) receiving nicotine and 3 of 33 patients (9%) of those on placebo.

Bandolier is not wholly convinced that these two trials measured the same outcome, but if these results are combined the relative benefit of transdermal nicotine over placebo was 2.6 (1.4 to 4.6) and the number needed to treat (NNT) was 3.7 (2.4 to 8.4). This means that of four patients with active ulcerative colitis who are given transdermal nicotine at around 15 - 22 mg/day for four to six weeks, one will have symptom improvement who would not have done so with placebo.



Comment

All the patients continued with their normal treatments during these trials. There are only two placebo-controlled studies, with only 66 patients given transdermal nicotine, and the confidence interval around the NNT includes an NNT of 8 - only half as good as the point estimate NNT of 3.7.

There were adverse effects of various types. These were common, and some patients could not tolerate nicotine patches. In the first study 10 of 35 patients given nicotine dropped out, for instance, and one patient given nicotine in the second study had acute pancreatitis.

Two other studies in the UK were not so positive. One [4] was a randomised comparison of transdermal nicotine with oral prednisolone in active ulcerative colitis for 6 weeks. Although 6 of 19 (32%) patients given nicotine achieved full sigmoidoscopic remission, so did 14/24 patients (41%) given oral prednisolone. Another study which compared transdermal nicotine (15 mg for 16 hours daily) with placebo in 80 patients with ulcerative colitis in remission showed no difference in relapses between the groups [5].

So transdermal nicotine may help some patients, but is no miracle cure for active ulcerative colitis, nor does it stop relapses in those patients in remission.

References:
1 MD Silverstein, BA Lashner, SB Hanauer. Cigarette smoking and ulcerative colitis: a case-control study. Mayo Clinic Proceedings 1994 69:425-9.
2 RD Pullan, J Rhodes, S Ganesh et al. Transdermal nicotine for active ulcerative colitis. New England Journal of Medicine 1994 330:811-5.
3 WJ Sandborn, WJ Tremaine, KP Offord et al. Transdermal nicotine for mildly to moderately active ulcerative colitis. Annals of Internal Medicine 1997 126:364-71.
4 GAO Thamas, J Rhodes, K Ragunath et al. Transdermal nicotine compared with oral prednisolone therapy for active ulcerative colitis. European Journal of Gastroenterology & Hepatology 1996 8:769-76.
5 GAO Thamas, J Rhodes, V Mani et al. Transdermal nicotine as maintenance therapy for ulcerative colitis. New England Journal of Medicine 1995 332:988-92.

NSAID-INDUCED GI INJURY

Bandolier 25 reported on the protective effects of misoprostol on NSAID-induced gastrointestinal (GI) bleeding shown in a large randomised study. It is useful to see those results largely confirmed in a superb meta-analysis of randomised trials of the ability of misoprostol and histamine antagonists to prevent NSAID-induced GI lesions [1]. The authors, from Rome, used techniques readers of ***Bandolier*** are familiar with, including L'Abbé plots, as well as giving NNTs for different levels of baseline risk from odds ratios, as demonstrated in ***Bandolier*** 36.

NSAID risks

Although NSAIDs are commonly prescribed by family physicians, their use triples the risk of developing severe GI adverse events. This meta-analysis assessed the effectiveness of misoprostol or histamine antagonists as preventive agents when coadministered with NSAIDs.

Systematic review

Clinical trials published between January 1970 through December 1994 were identified by searching both MEDLINE and relevant review articles. Inclusion criteria were:

- endoscopy performed before NSAID treatment
- no evidence of ulcer initially
- random allocation with a placebo arm
- NSAIDs given for at least 5 days

A variety of NSAIDs were used and drug dosages were not specified. Histamine antagonists studied were ranitidine, cimetidine and nizatidine. Daily misoprostol dosages ranged from 200-800 micrograms. Outcomes were examined with respect to short term (< 2 weeks) or long term (>4 weeks) exposure to NSAIDs. The main endpoints measured were the number of subjects in which gastric ulcers, gastric lesions (> 5 erosions or 1 ulcer), duodenal ulcers, and duodenal lesions appeared. Ulcer disease is the key outcome, as the clinical significance and natural history of erosions is unclear.

Data for analysis

The search found 24 articles describing 4325 patients. About 16 percent were volunteers, and the rest were patients with acute musculoskeletal disease, osteoarthritis or rheumatoid arthritis. Analysis was by rate difference between patients on NSAIDs treated with histamine antagonist or placebo.

Results

Gastric ulcers

The weighted average baseline risks for gastric ulcers were 3.6% and 6.8% with <2 weeks and >4 weeks use of NSAIDs. For gastric ***lesions*** the weighted average baseline risks were 53% and 27% respectively.

Misoprostol use significantly reduced the rate of gastric ulcers both in short-term (rate difference -13%; 95%CI, -26% to -1%) and long-term (rate difference -8%; 95% CI, -18% to -1%) NSAID treatment. This translates into NNTs of 8 for <2 weeks treatment and 12.5 for >4 weeks treatment. Histamine antagonists were not found to reduce the rate of gastric ulcers with either short term or long term trials.

Duodenal ulcers

The weighted average baseline risks for duodenal ulcers were 3.0% and 4.0% with <2 weeks and >4 weeks use of NSAIDs. For duodenal ***lesions*** the weighted average baseline risks were 11% and 12% respectively.

Both histamine antagonists and misoprostol significantly reduced the risk in the long term trials, but neither result was clinically significant (NNT = 42 and 29, respectively), and neither agent had an effect on short term trials.

Results were not modified significantly whether normal subjects or patients were studied, although there were no stud-

Effect of baseline risk on misoprostol prophylaxis for NSAID-induced gastric ulcer over short and long term

Short-term prophylaxis NNT (95%CI)	Baseline risk (%)	Long-term prophylaxis NNT(95%CI)
35 (34 - 39)	3	47 (41 - 58)
21 (20 - 23	5	29 (25 - 35)
11 (10 - 12)	10	15 (13 - 18)
5 (5 - 6)	20	8 (7 - 10)
4 (3 - 4)	30	5 (5 - 7)
3	40	5 (4 - 7)

Odds ratio for short term protection with misoprostol 0.06 (0.03 - 0.15) for short-term (<2 weeks) and 0.29 (0.20 - 0.42) for long-term (>4 weeks) co-administration with NSAID in subjects without ulcers.

ies of long term prevention with normal subjects. Studies with misoprostol had patients with a higher risk in the control groups than with histamine antagonist comparisons. The authors addressed this by calculating NNTs with baseline risks between 3 and 40% for gastric ulcer. For instance, with a baseline risk of 3% the NNT for misoprostol to prevent one ulcer over two weeks of NSAID exposure compared with placebo was 35. At a baseline risk of 40% it was 3.

Comment

This study was an overview of *lesion* prevention. Lesions are not the same as clinically significant GI bleeding caused by NSAIDs. The large randomised study which had clinical effects as an outcome [2] indicated that misoprostol use may be usefully considered in patients at high risk for complications on NSAID therapy - specifically, patients older than 75 years of age or with a history of peptic ulcer disease or upper GI tract bleeding. The table of NNTs with differing baseline risk emphasises this advice. The drugs should be coadministered as soon as possible, because the risk of erosive lesions is high from the start of NSAID use.

References:
1 Koch M, Dezi A, Ferrario F, Capurso L. Prevention of nonsteroidal anti-inflammatory drug-induced gastrointestinal mucosal injury. Archives of Internal Medicine 1996 156: 2321-32.
2 FE Silverstein, DY Graham, JR Senior et al. Misoprostol reduces serious gastrointestinal complications in patients with rheumatoid arthritis receiving nonsteroidal anti-inflammatory drugs. Annals of Internal Medicine 1995 123: 241-9.

THROMBOPROPHYLAXIS AND DEATH AFTER TOTAL HIP REPLACEMENT

Bandolier has visited the issue of thromboprophylaxis or compression stockings after surgery before (***Bandolier*** 17, 18). A new meta-analysis [1] asks and answers the question of what is the risk of fatal pulmonary embolism and total mortality after total hip replacement (THR) and to what extent do different methods of thromboprophylaxis affect mortality?

Background

Studies conducted in the 1960s found an incidence of fatal pulmonary embolism after THR of approximately 1%, and authoritative sources currently recommend thromboprophylaxis for patients undergoing THR. Many deep venous thromboses (DVTs) detected in trials are clinically asymptomatic, and DVTs are used as surrogate end-points because they can be measured and because they occur commonly (40% to 80% of cases). Many studies have shown a venographic reduction in the risk of DVT with various prophylactic agents (heparin, warfarin, aspirin, etc.). The assumption inherent in these investigations is that DVT sometimes causes pulmonary embolism which, at times, results in death.

Meta-analysis

A MEDLINE search (from 1966-95) was conducted to find all the relevant literature that discussed fatal pulmonary embolism and total mortality in patients receiving THR. It was not restricted to prospective randomised controlled trials, but included all studies of THR in which the number of patients being studied and total number of deaths or fatal pulmonary embolisms were reported.

Types of thromboprophylaxis were broken down into six categories:

1 None (including patients who received placebo, compression stockings, or no prophylaxis)
2 Heparin (including low molecular weight and unfractionated),
3 Warfarin,
4 Aspirin,
5 Dextran,
6 Other (usually a combination of medications)

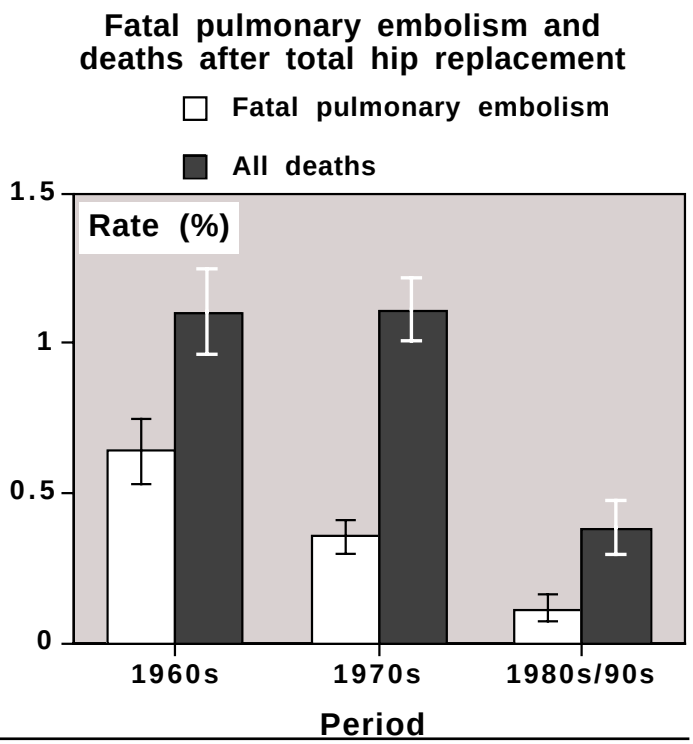
Patients receiving active prophylaxis may have also worn compression stockings. The results of this heterogeneous mix of studies were combined with appropriate meta-analytic techniques.

Outcomes

The primary outcomes were the rates of fatal pulmonary embolism and total mortality after total hip replacement.

Results

About 93,000 patients in 181 papers were analysed. Over the last 15 years, the rate of fatal pulmonary embolism was 0.11% (95%CI 0.07% to 0.16%) and total mortality was 0.38% (95%CI 0.29% to 0.47%). These rates had declined dramatically from 0.64% for fatal pulmonary embolism and 1.1% for overall mortality in the 1960s, probably because of better techniques.



No differences emerged in the efficacy of various thromboprophylaxis regimens preventing fatal pulmonary embolism. This was primarily due to the very low incidence of this outcome. For example, only 4 fatal pulmonary embolisms were reported in 3432 patients (0.12%) who did not receive prophylaxis and only 8 fatal pulmonary embolisms were reported in 10,356 patients (0.08%) who received heparin. Overall mortality did not differ between any of the prophylaxis regimens or when they were compared with no prophylaxis.

Prophylaxis	95%CI of fatal pulmonary embolism rate (%)
None	0.03 to 0.30
Heparin	0.03 to 0.15
Warfarin	0.00 to 0.14
Aspirin	0.02 to 0.32
Dextran	0.10 to 0.53

Comment

This meta-analysis challenges the current dogma regarding thromboprophylaxis in patients receiving THR. The summary suggests that because there is not enough evidence in the literature to conclude that any form of prophylaxis decreases the death rate after THR, "guidelines which recommend their routine use to prevent death after hip replacement are not justified".

This is strong stuff. The paper indicates that thromboprophylaxis is unlikely to reduce the rate of fatal PE after THR by more than 0.05% (that is, 5 in 10,000). If the death rate from complications of thromboprophylaxis (bleeding, for instance) is greater than 5 in 10,000, then thromboprophylaxis may cause more harm than benefit. The risk of clinically significant thrombotic events caused by heparin-induced thrombocytopenia is about 3% in patients receiving unfractionated heparin [2].

It would be helpful to quantify the harm resulting from thromboprophylaxis (probably though new systematic reviews looking specifically at harm), because randomised trials of sufficient size to measure the low rates of death and benefit would be huge, expensive, and take a long time.

References:
1 DW Murray, AR Britton, CJK Bulstrode. Thromboprophylaxis and death after total hip replacement. Journal of Bone Joint Surgery (Br)1996 76-B: 863-70.
2 TE Warkentin, MN Levine, J Hirsh, et al. Heparin-induced thrombocytopenia in patients treated with low-molecular weight heparin or unfractionated heparin. New England Journal of Medicine 1995 332: 1330-5.

Old Curiosity Shop

Thanks to those who have sent in suggestions. ***Bandolier*** has chosen a paper published 25 years ago which should be re-read. It examines US attitudes to mental health, which have most likely changed, and though it is not directly relevant to today's UK practice, the philosophy keeps feet firmly on the ground.

On being sane in insane places

Yes, we all feel like this at some time or another. But the study in this paper from Stanford [1] was a bit different. Eight sane people (a varied group, from a psychology graduate in his '20s, three psychologists, a paediatrician, a psychiatrist, a painter and a housewife) gained admission to 12 psychiatric hospitals in five US states on the East and West coasts. They all alleged "pseudosymptoms" and those in mental health professions pretended to have other jobs. Their presence was not known to hospital staff.

Not one was spotted

Basically the pseudopatients arranged a hospital appointment, complained of hearing voices, but made no other significant change to their life history. All were admitted, except in one case, with a diagnosis of schizophrenia.

Immediately on admission the pseudopatients ceased simulating ***any*** symptoms, and apart from some understandable nervousness, behaved normally. They did make copious notes on the ward about what happened.

Despite their public show of sanity, the pseudopatients were never detected by medical or nursing staff, and were discharged (after between 7 and 52 days, average stay 19 days) with a diagnosis of schizophrenia "in remission". Fellow patients were better at spotting the pseudopatients - 35 of 118 fellow patients voiced suspicions about the pseudopatients.

It couldn't happen here!

Two hospitals which had heard of the findings doubted that such an error could happen there. So staff were informed that over the coming three months one or more pseudopatients would attempt to be admitted. Each staff member was asked to rate each patient presenting at admission according to the likelihood of that patient being a pseudopatient.

Judgements were obtained on 193 patients:

- 41 patients (21%) were alleged, with high confidence, to be pseudopatients by at least one member of staff.
- 23 patients (12%) were considered suspect by at least one psychiatrist.
- 19 patients (10%) were considered suspect by one psychiatrist and one other staff member.

No pseudopatient actually presented during this time.

Sobering thoughts

The results are sobering enough. Reading the extensive commentary about the details of treatment and process is chilling. It certainly make one think about diagnostic accuracy (as ***Bandolier*** has pointed out in issue 37 for malignant melanoma). The message about how to treat people is one that even a caring service like the NHS should remember from time to time.

Reference:
1 DL Rosenhan. On being sane in insane places. Science 1973 179:250-8.

'EVIDENCE-BASED HEALTH CARE'

David Sackett's definition of 'evidence based medicine' (EBM) is now well known and widely accepted. But the phrase 'evidence based health care' (EBHC) is rarely defined. Much of my work involves explaining and trying to apply the principles of EBM and EBHC, often to people who have been puzzled and even irritated by what they had thought EBM and EBHC implied about their current and past practice. I have evolved my explanation of EBHC into a definition:

"Evidence based health care takes place when decisions that affect the care of patients are taken with due weight accorded to all valid, relevant information."

Several things follow from this definition:

1 **'decisions that affect the care of patients'** are taken by managers and health policy makers as well as by clinicians. EBHC is therefore just as relevant to managers and policy makers as it is to clinicians.

2 **'due weight'** implicitly acknowledges that there are many factors that contribute to decisions about the care of patients. There are many factors other than the results of randomised controlled trials that may weigh heavily in both clinical and policy decisions (for instance, patient preferences and resources). This definition requires that valid, relevant evidence should be considered alongside other relevant factors in the decision making process. It does not assume that any one sort of evidence should necessarily be the determining factor in a decision.

3 **'all'** is aspirational - but it implies that there should be an active search for valid, relevant information

4 **'valid, relevant'** implies that before information is used in a decision, an assessment should be made of the accuracy of the information and the applicability of the evidence to the decision in question; that is, information should be appraised.

5 **'information'** is deliberately left unspecified; there are many types of information that may be valid and relevant in particular circumstances. I have no wish to exclude any particular type of information as long as an appraisal is made of its validity and relevance and the information is given 'due weight' - neither more nor less.

Other things follow from this definition, not least that the concept of EBHC is not new - it's what most people I know have been trying to practice all their working lives. But there are new reasons and new opportunities to help us improve the care that patients receive including:

a) more and better information e.g. from the increasing number of well conducted RCTs and systematic reviews,
b) the better organisation of information and new insights that derive from the evolving science of systematic review,
c) rapid advances in information technology and
d) an improving (though still inadequate) understanding of the (social and organisational) processes by which research findings are translated into practice.

I have been using this definition for a year or so now. People seem to find it useful and non-threatening. I would value the thoughts and any suggestions for improving it from readers of ***Bandolier***.

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BANDOLIER 2ND ANNUAL

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